SPINAL INSTABILITY SECONDARY TO METASTATIC CANCER

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Fifty-five patients with severe pain from spinal instability secondary to metastatic cancer were referred to Hope Hospital, none being judged to be in a terminal condition. One patient had too extensive disease for surgery so 54 were treated by 55 spinal stabilisations; 49 obtained complete relief of pain and two had partial relief. There were three failures.

Twenty-eight of the patients had clinical evidence of spinal cord or cauda equina compression and were decompressed at the time of stabilisation. Of these, 20 had major recovery of neurological function. Patients with pre-operative evidence of extradural tumour had 'prophylactic' decompression at the time of stabilisation; none of these patients later developed signs of cord or cauda equina compression.

The results suggest that alleviation of pain and restoration of mobility are best achieved by segmental spinal stabilisation; a few patients require a combined anterior and posterior stabilisation. Postoperative radiotherapy should be given whenever possible, and the causative tumour should be treated by endocrine or chemotherapy, as indicated.

Back pain is common in patients with disseminated carcinoma; in about 10% of them this is attributable to spinal instability (Galasko and Sylvester 1978). This can produce excruciating pain that is mechanical in nature, and when it is severe the patient is only comfortable when lying absolutely still. Any movement, even rolling by trained nurses, causes very acute pain; the patient may not be able to sit, stand or walk, even with the use of a spinal orthosis. In milder cases the patient may be relatively free from pain when wearing a rigid spinal support, but movement of the back, turning in bed, sitting or standing may be impossible without the orthosis.

In such patients, radiographs show destruction of bone with vertebral collapse. No discrete fracture can be seen, but the secondary spinal instability should be regarded as the equivalent of a pathological fracture in a long bone. Pain is caused by the instability and not by the metastasis, and is not alleviated by radiotherapy or chemotherapy. As in other pathological fractures, immobilisation is required for pain relief.

The management and results in 55 patients with severe thoracic or lumbar instability secondary to spinal metastases are reported.

PATIENTS AND METHODS

All 55 patients presented with a history of malignancy and severe pain in the dorsal or lumbar spine which was mechanical in nature and relieved by lying absolutely still. Almost all were referred by oncologists or radiotherapists, chemotherapy and irradiation having failed to control their pain. Patients with milder forms of spinal instability, who were successfully managed in spinal orthoses, were excluded from the study as were patients with instability of the cervical spine.

Patients had full clinical and neurological examination, chest radiography, full blood counts and biochemical profiles including serum calcium. Anteroposterior and lateral radiographs were taken of dorsal and lumbar spines. When possible, pre-operative skeletal scintigraphy was used to evaluate the extent of skeletal dissemination, but urgent surgery was not delayed for scintigraphy. All, except the first patient, had pre-operative myelography; combined CT scanning was also routine once it became available. The first patient had no clinical evidence of cord compression and a very successful result after stabilisation with two Harrington rods. Eight months later he developed cord compression and died three weeks later despite emergency decompres-
sion which required removal of the rods. This experience led to the routine use of pre-operative myelography for every patient, with decompression if there was any evidence of extradural tumour.

Operation. Several techniques of spinal stabilisation were used without concomitant bone grafting (Table I). The aim was to provide stability, restore function and painfree mobility, and to obviate the need for an external support. Initially Harrington rods were used since they were the only implants that were available. Two distraction rods had their fixation supplemented by methylmethacrylate around the hooks.

The Banks–Dervin rod, used later, depends on screw fixation at multiple levels. The rod is embedded in the gutter between the spinous process and one lamina; the screw is passed through the base of the spinous process into the contralateral lamina (Fig. 1). The rod can be moulded to the shape of the spine by special bending instruments. It is 1 cm square and is so rigid that only a single rod is required (Banks and Dervin 1980). It is usually placed on a thin bed of methylmethacrylate to eliminate any space between the rod and the vertebral column.

The Luque ‘L’ rods and the Hartshill rectangle are fixed to the spine by sublaminar wires. The Banks–Dervin rod, Luque L rods and Hartshill rectangle were fixed to two or preferably three vertebrae above and below the unstable segments. If the radiographs indicated that there were two areas of instability the stabilisation supported both areas. Where the CT scan indicated that the quality of bone stock in the adjacent vertebrae was poor or skeletal scintigraphy indicated that the adjacent vertebrae were substantially affected by metastatic disease, the stabilisation was extended over a larger area so that the implant was fixed to unaffected vertebrae (Fig. 2). When the stabilisation extended to the sacrum, a lumbar lordosis was moulded into the rod or rectangle to prevent a flat back deformity that might have interfered with comfortable sitting.

In two patients, posterior stabilisation alone was not considered to be adequate, because the level of instability was at L5 or L4. These patients had two-stage combined anterior and posterior stabilisation. In the first stage the dura was decompressed anteriorly and the spine stabilised with methylmethacrylate; at the second stage a moulded Hartshill rectangle was used posteriorly.

If the patient had any signs or symptoms suggestive of compression of the spinal cord or cauda equina or if compression was seen on the pre-operative myelogram, decompression was performed at the time of spinal stabilisation. This involved removal of both laminae and of the spinous process at the level or levels of compression.

Postoperative management. The patients were all referred for consideration of postoperative irradiation; this was not always feasible since some of them had received a maximum dose before surgery, and further radiation might have compromised the cord. Depending on the primary lesion, some patients were also considered for postoperative chemotherapy or endocrine therapy.

RESULTS

None of the 55 patients was thought to be in a terminal condition, that is likely to die from cancer within a few days. No patients had malignant hypercalcaemia. The commonest primary tumour was carcinoma of the breast but a very wide variety of tumours can metastasise and result in spinal instability (Table II). One patient had such gross destruction of her dorsal and lumbar spine that surgical stabilisation was judged not to be feasible. She had some relief from an orthosis, but was unable to move or even roll in bed without it. The other 54 patients underwent 55 spinal stabilisations, one having lumbar instability treated 22 months after a successful dorsal stabilisation.

An outline of the results and complications is given...
in Table III. Of the five patients treated with Harrington rods, only one survived for more than nine months and in this patient the rod loosened and was replaced with a Banks–Dervin rod. The other Harrington rods would probably have loosened had the patients survived for longer, and it became obvious that some form of segmental stabilisation was required.

The Banks–Dervin rod was specifically designed for use in metastatic disease of the spine (Banks and Dervin 1980), but since the Hartshill rectangle was developed the latter has been used in most patients. Both the Banks–Dervin rod and the Hartshill rectangle provided sufficient stability to avoid the postoperative use of spinal orthoses and to allow posterior decompression at the time of stabilisation.

One Banks–Dervin rod was removed because of deep infection; one Harrington and one Banks–Dervin rod became loose. The Banks–Dervin rod loosened because of inadequate proximal fixation. Two patients obtained partial relief of pain; complete relief was achieved in 49 of 55 operations (89%). Wound infection in one patient was successfully treated with antibiotics...
without removal of the implant. The wound broke down in one patient who had had extensive pre-operative irradiation; it healed after secondary suture.

Double Luque L rods with segmental sublaminar wire fixation were used in only one case. One of the rods broke after 18 months (Fig. 3) and the patient needed subsequent anterior bone grafting. The patient is still alive five years after the initial operation.

One 5 mm Hartshill rectangle broke five years after insertion (Fig. 4). Fortunately, the patient had responded well to endocrine therapy and had some reconsolidation of her vertebral column. The pain that developed after implant failure was controlled by an orthosis.

In 28 patients there was some clinical evidence of compression of the spinal cord or cauda equina, usually weakness of the lower limbs severe enough to affect walking or standing. Weakness usually preceded the development of bladder paralysis. These patients were treated by decompression at the time of stabilisation; 20 of the 28 (71%) obtained major recovery of neurological function, sufficient to allow them to walk without aids and to restore bladder function where it had been

Table II. Primary tumours in 55 patients with spinal instability due to metastases

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Number</th>
<th>Tumour</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>31*</td>
<td>Cervix</td>
<td>1</td>
</tr>
<tr>
<td>Meloma</td>
<td>8</td>
<td>Vagina</td>
<td>1</td>
</tr>
<tr>
<td>Prostate</td>
<td>2</td>
<td>Stomach</td>
<td>1</td>
</tr>
<tr>
<td>Melanoma</td>
<td>2</td>
<td>Parotid</td>
<td>1</td>
</tr>
<tr>
<td>Bronchus</td>
<td>1</td>
<td>Lymphoma</td>
<td>1</td>
</tr>
<tr>
<td>Kidney</td>
<td>1</td>
<td>Chondrosarcoma</td>
<td>1</td>
</tr>
<tr>
<td>Colon</td>
<td>1</td>
<td>Histiocytoma</td>
<td>1</td>
</tr>
<tr>
<td>Uterus</td>
<td>1</td>
<td>Cordoma</td>
<td>1</td>
</tr>
</tbody>
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* one patient had disease which was too extensive for stabilisation and one patient had a second stabilisation at another level (see text)

Table III. Results and complications in 54 patients who had operations for spinal instability due to metastases

<table>
<thead>
<tr>
<th>Result</th>
<th>Number</th>
</tr>
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<tbody>
<tr>
<td>Relief of pain</td>
<td></td>
</tr>
<tr>
<td>complete</td>
<td>49</td>
</tr>
<tr>
<td>partial</td>
<td>2</td>
</tr>
<tr>
<td>Loosening of implant</td>
<td>2</td>
</tr>
<tr>
<td>Infection resulting in removal of implant</td>
<td>1</td>
</tr>
<tr>
<td>Complications*</td>
<td></td>
</tr>
<tr>
<td>infection treated successfully</td>
<td>1</td>
</tr>
<tr>
<td>wound breakdown, successful secondary suture</td>
<td>1</td>
</tr>
<tr>
<td>fracture of a Luque L rod requiring additional anterior stabilisation</td>
<td>1</td>
</tr>
<tr>
<td>fracture of a Hartshill rectangle (at five years)</td>
<td>1</td>
</tr>
</tbody>
</table>

* in the 51 patients with relief of pain

Figure 4a – A patient with mammary carcinoma presented with severe pain and substantial weakness affecting her left leg. The radiculogram shows destruction of L3 with a large epidural mass. Figures 4b and 4c – Radiographs taken two years after the initial operation for posterior decompression and stabilisation with a Hartshill rectangle. The patient had responded well to chemotherapy and there is substantial reconsolidation of L3. Figure 4d – Radiograph showing further destruction of L3 with fracture of the Hartshill rectangle. Low back pain was easily controlled by a lightweight orthosis and endocrine therapy was modified. The patient regained full leg function with no clinical recurrence of neurological involvement.

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compromised. In two patients cord compression recurred 10 and 14 months, respectively, after decompression.

The first 29 patients have been followed-up until death. Their average survival was 36 weeks, but one patient lived for three and a half years and one for four years. Many of the later patients are still alive, some at five to six years after surgery.

**DISCUSSION**

The development of spinal instability is not necessarily a terminal event. With improvements in endocrine treatment and chemotherapy patients will probably live longer, as they do with pathological fractures of the long bones. Therefore it is not surprising that some patients are alive and well five and six years after stabilisation. In most patients, instability of the dorsal or lumbar spine secondary to metastatic cancer can be adequately treated by posterior stabilisation alone, with some form of segmental fixation, as also reported by others (Flitieley, Anderson and Anast 1984; DeWald et al 1985). Infection developed in two of DeWald et al’s 17 patients but both had immunosuppression as a result of pre-operative chemotherapy.

The implant should be fixed to at least two and preferably three vertebrae above and three vertebrae below the area of instability. Pre-operative scintigraphy and CT myelography help to demonstrate which vertebrae have sufficient bony stock to allow fixation of the implant. If the level of the instability does not allow adequate posterior stabilisation, as at L4 or L5, combined anterior stabilisation is needed.

The two implants that failed were double Luque L rods and a 5 mm Hartshill rectangle: a Banks–Dervin rod or a 6 mm Hartshill rectangle should be used. There were no failures with either of these implants even though bone grafting and postoperative orthoses were not used.

It has been suggested that the optimum treatment is anterior stabilisation, since metastases usually arise in the vertebral body (Harrington 1981; Boland, Lane and Sundaresan 1982; Siegel, Tiqva and Siegal 1985; Turner et al 1988). However, these authors reported patients with extradural tumours of the spine mostly presenting with neurological symptoms as a result of cord compression. Most of the operations were for spinal decompression; in several instances they were late.

None of the patients in this series presented primarily with cord or cauda equina compression, but rather with pain secondary to spinal instability. There was an associated neurological deficit in 28 of the patients, but in all cases the compression was early – none had complete paraplegia or urinary retention of more than 18 hours’ duration at the time of operation. In these patients tumour tissue had completely surrounded the cord and could be seen posterior to the dura at the decompression operation. Where the tumour has completely surrounded the spinal cord or cauda equina, it does not seem to matter whether the spinal canal is decompressed anteriorly or posteriorly, provided that the decompression is adequate and completely relieves the pressure on the neural elements within the canal. However, for late cases of cord compression, anterior decompression might offer the only chance of bladder recovery.

Whenever possible after stabilisation, the lesion should be irradiated and the patient given chemotherapy or endocrine therapy to prevent further growth of the tumour. Despite such treatment the tumour was not always controlled: two patients had evidence of recurrent cord compression 10 and 14 months after successful decompression.

**Conclusions.** Posterior stabilisation is at least as effective in the control of spinal instability as an anterior approach; with local decompression it is equally useful in the treatment of early cord or cauda equina compression.

Patients with spinal instability should have pre-operative myelography or CT scanning or both. Evidence of cord or cauda equina compression is an indication for decompression at the time of stabilisation. Posterior decompression appears to be adequate since no patient with radiographic evidence of extradural tumour, but without clinical symptoms of compression, subsequently developed any sensory, motor or bladder symptoms.

Where there is neural compression without instability, surgical decompression should be combined with prophylactic stabilisation.

Pre-operative irradiation should be avoided since it may be associated with increased bleeding, delayed healing or wound breakdown and infection. Postoperative radiotherapy should be given whenever feasible, and the causative tumour should be treated by endocrine or chemotherapy as indicated.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


